accelerate the age-related RILA stiffness occurs earlier than to accelerate the age-related RAA stiffness. The mechanism is probably associated with the up-regulated level of RAGE in IRSA media, while the AGEs in serum or IRSA media may be involved in the late stage.

GW26-e4656

Polymorphism of RBP4 Locus Is Associated with 5-Year Survival in acute coronary syndrome after coronary revascularization

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OBJECTIVES The rs7094671 was single nucleotide polymorphism of RBP4 locus that was associated with prevalence of coronary artery disease. No data concerning their association with long term prognosis after myocardial infarction is available. The aim of our study was to investigate the association of the RBP4 locus with 5-year overall mortality in patients with acute coronary syndrome after coronary revascularization.

METHODS Cohort study included 292 patients with acute coronary syndrome treated with primary PCI, followed for up to 5 years. Genotyping was performed with high resolution melting (HRM) analysis. The analyzed end-point was total 5-year mortality.

RESULTS The baseline characteristics were well-balanced between carriers (AA, n = 19; AG, n = 73) and GG (n = 200) of the RBP4 variant. During the follow-up period (52.45±11.98months), the primary endpoint occurred more frequently in carriers of A allele than in non-carriers of A allele (24.8% versus 9.3%; hazard ratio [HR] = 2.656; 95% confidence interval [CI] = 1.642-4.295; P = 0.000); Kaplan-Meier estimation of the primary outcome measure (death of any cause) during the follow-up period. (AA groups versus GG groups: adjusted HR = 6.321, 95% CI 2.081-19.205, P = 0.001), GA groups versus GG groups: adjusted HR = 1.303, 95% CI = 0.478-3.548, P = 0.605).

CONCLUSIONS The RBP4 locus is associated with 5-year mortality in high-risk patients with acute coronary syndrome.

GW26-e4667

Effect of Oxidatively Modified Low-Density Lipoprotein on Osteodifferentiation of Mesenchymal Stem Cells Co-cultured with Vascular Smooth Muscle Cells

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OBJECTIVES Increasing evidences revealed that bone marrow-derived mesenchymal stem cells (BM-MSCs) played important role in wound healing and vascular remodeling in vivo. However, the mechanism in the development of atherosclerosis and vessel calcify remains unclear. The aim of this study was to investigate the effect of oxidatively modified low-density lipoprotein (ox-LDL) on osteodiferenliation of BM-MSCs co-cultured with smooth muscle cells with or without osteogenic inductor, and further to explore the mechanism of BM-MSCs participating in atherosclerosis and vessel calcify.

METHODS BM-MSCs and vascular smooth muscle cells (VSMCs) were prepared from Sprague-Dawley rats and co-cultured in a transwell coculture system, which allowed the diffusion of secreted factors but prevented cell contact. Thecombined group (both osteogenic inductor and ox-LDL), ox-LDL group, osteogenic inductor group and control group were allocated according to factorial design method. The effect of ox-LDL on the osteogenic potential was determined by cell morphology, real-time PCR, immunofluorescent staining, alkaline phosphatase (AKP) activity and osteopontin (OPN) synthesis.

RESULTS 1. All groups expressed OPN mRNA and AKP and but the OPN mRNA and AKP expression levels of combined group were the highest after 7 days and 10 days of cell culture, moreover, there was positive interaction between osteogenic inductor and ox-LDL ($P < 0.01$).

2. The result of immunity histochemistry also showed that OPN and AKP expression levels in combined group were the highest after 14 days of cell culture.

CONCLUSIONS Ox-LDL can promote osteogenic inductor-mediated osteodifferentiation of BM-MSCs co-cultured with smooth muscle cells.

GW26-e4724

The role of mAKAPβ in the process of cardiomyocyte hypertrophy induced by angiotensin II

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OBJECTIVES Angiotensin II (AngII) is the central product of the reninangiotensin system (RAS) and this octapeptide contributes to the pathophysiology of cardiac hypertrophy and remodeling. mAKAPβ is an Akinase anchoring protein (AKAP) that has the function of binding to the regulatory subunit of protein kinase A (PKA) and confining the holoenzyme to discrete locations within the cell. In this study, we aimed to investigate the role of mAKAPβ in AngII induced cardiomyocyte hypertrophy and the possible mechanisms involved.

METHODS Cultured cardiomyocytes from neonatal rats were treated with AngII. Subsequently, the morphology of the cardiomyocytes was observed and the expression of mAKAPβ and cardiomyocyte hypertrophic markers was measured. mAKAPβ-shRNA was constructed for RNA interference; the expression of mAKAPβ and hypertrophic markers, the cell surface area and the [3H] Leucine incorporation rate in the AngII-treated rat cardiomyocytes were detected following RNA interference. Simultaneously, changes in the expression levels of phosphorylated extracellular signal-regulated kinase (p-ERK)2 in the cardiomyocytes were assessed.

RESULTS The cell size of the AngII - treated cardiaomyocytes was significantly larger than that of the untreated cardiomyocytes. The expression of hypertrophic markers and p-ERK2, the cell surface area and the [3H] Leucine incorporation rate were all significantly increased in the AngII - treated cells. However, the expression of mAKAPβ remained unaltered in this process. RNA interference simultaneously inhibited the protein expression of mAKAPβ and p-ERK2, and the hypertrophy of the cardiomyocytes induced by AngII was attenuated.

CONCLUSIONS AngII induces hypertrophy in cardiomyocytes and mAKAPβ is possibly involved in this process. The effects of mAKAPβ on AngII-induced cardiomyocyte hypertrophy may be associated with p-ERK2 expression.

GW26-e4732

The Effect Of Catecholamine Release-Inhibitory Peptide Catestatin On Heart Rate And Blood Pressure Of Hypertension

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OBJECTIVES The catecholamine release-inhibitory peptide catestatin is an endogenous nicotinic cholinergic antagonist that can inhibit secretion of catecholamine from chromaffin cells and adrenergic neurons. Therefore it can inhibit sympathetic nerve activity. Previous studies have shown that catestatin have a close relationship with the occurrence and development of hypertension and that catestatin is diminished in established hypertension. This study focus on the effect of catestatin on heart rate and blood pressure of hypertension by supplementing spontaneously hypertensive rats (spontaneously hypertensive rats, SHR) with catestatin.

METHODS We used SHR as a hypertensive model and matched it with the homologous normotensive rats (wistar-Kyoto, WKY) as normal control group. Blood pressure and heart rate were obtained from the measurement tail-cuff blood pressure. First, we observed the difference of blood pressure and heart rate between SHR and WKY at the age of 6, 12 and 16 weeks. Second, twelve-week-old SHR were treated with 1mg catestatin via the tail vein injection. Thirty minutes after injection, heart rate and blood pressure were measured to observe its short-term effects on blood pressure and heart rate. Furthermore, we also treated twelve-week-old SHR with catestatin for 5 weeks (tail vein injection for three times a week, each 1mg) to observe its long-term effects on blood pressure and heart rate. Each group has 5 rats.

RESULTS First, the blood pressure and heart rate of SHR were significantly higher than that of WKY at corresponding ages. SHR *VS* WKY, six-week-old: heart rate (418±28 *VS* 366±24 (beats/min), P<0.05), blood pressure (148±9/108±6 *VS* 118±11/85±9mmHg, P<0.05); twelve-week-old: heart rate (397±30 *VS* 326±25 (beats/min), P<0.05); blood pressure (193±15/155±12 *VS* 123±7/91±10 mmHg,